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Defect-induced anticorrelations in molecular motor traffic

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Abstract

We revisit the nonequilibrium phase transition between a spatially homogeneous low-density phase and a phase-separated high-density state in the deterministic sublattice totally asymmetric simple exclusion process with stochastic defect. We discuss this phase transition in a grandcanonical ensemble for which we obtain exact results for the stationary current-density correlations and for the average collective velocity. We identify defect-induced anticorrelations that are absent in similar boundary-induced phase transitions. The average collective velocity vanishes at the phase transition and in the phase-separated state due to its macroscopic spatial inhomogeneity.

Keywords: driven diffusive systems, molecular motors, totally asymmetric simple exclusion process with blockage, nonequilibrium steady state, current-density correlations

1. Introduction

There is a large variety of molecular motors that move in a cell along a template or track while performing some biological function. Examples include ribosomes moving along mRNA template for protein synthesis, RNA polymerases moving along DNA, or kinesins and other motor proteins moving along microtubules for cargo transport [1]. Exploring the mechanical aspects of this motion from a biophysics perspective sheds light on common features of the kinetics of such molecular motor 'traffic' [2] and on collective phenomena arising from the interactions between motors performing their task simultaneously on the same track [3–8]. To further highlight the significance of investigating molecular motor traffic we mention that from

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a medical viewpoint this research is important among other things for investigating the causes of various diseases including Alzheimer's (related to kinesin motors [9, 10]) or AIDS (related to programmed frameshift of RNA polymerase)) [11–13], and for understanding changes in antibiotic resistance [14, 15] due to synonymous mutation of the DNA sequence.

Two common features of molecular motor traffic are (i) translocation of the motors along their track—which is a quasi one-dimensional step-wise driven motion from one binding site to the next—and (ii) steric hindrance between motors due to excluded volume. These properties lend themselves to theoretical analysis by one-dimensional asymmetric exclusion processes (ASEPs) [16, 17] which are driven particle systems where translocation is modelled by directed particle jumps along a one-dimensional lattice and steric hindrance is taken into account by the exclusion principle that forbids occupancy of the same lattice site by more than one particle, see [2, 7, 8] for extensive surveys and [18] for some recent developments.

A further frequent feature is the appearance of inhomogeneities along the track that lead to a significant slowing down of translocation at specific binding sites [19].[1] In the ASEP approach such slow sites can be modelled by defects with a significantly reduced jump probability [25–30]. The interplay of directed translocation, steric hindrance and the presence of a defect is known to lead to 'molecular traffic jams' [2] with strong impact on protein production rates, efficiency of cargo transport or other cellular mechanisms associated with molecular motors.

Localized defects in driven particle systems have a long history of study also from a statistical physics perspective [31–37] and they continue to intrigue [38–42]. Generally, at a critical motor density ρ_c along the track there is a phase transition from a spatially homogeneous free flow state for $\rho < \rho_c$ to a phase-separated congested state for $\rho > \rho_c$ where the stationary particle current becomes independent of the conserved particle density. The phase-separated state arises from the formation of a macroscopic 'traffic jam' behind the defect, consisting of a congested high density domain and separated by a microscopically sharp domain wall from the free-flow low-density domain [43–45]. Thus this phenomenon can be regarded as a nonequilibrium analog of phase separation [46, 47]. The domain wall performs a diffusive random motion with vanishing mean velocity at the critical point, see [48–53] for mathematically rigorous results for exclusion processes.

Also the significance of correlations in active biological transport has been discussed using the lattice gas approach [54–56]. In this work we study correlations between translocation at some binding site and the presence of motors on some other–possibly far distant—binding site. In particular, we discuss (i) the emergence of anticorrelations that have been discovered recently also for the particle density [57, 58] and (ii) how the phase transition is reflected in the velocity of kinematic density waves that arise as a collective phenomenon from local density perturbations [17, 59–62].

The paper is organized as follows. In section 2 the lattice gas model is defined and known stationary properties relevant for the present work are reviewed. In section 3 we derive and discuss the exact stationary current-density correlation function and in section 4 the collective motor velocity is obtained. In section 5 we present some conclusions. Technical details of the mathematical computations are given in the appendices.

[1] In the context of the diseases mentioned above such 'slow sites' may arise from structural imperfections of the microtubular structure [20], pseudoknots on mRNA templates [11, 21], or synonymous mutation of the DNA sequence [22–24].

2. The dsTASEP with a slow site

For self-containedness we recall the precise definition of the process and some of its basic stationary properties which were proved in [33, 57, 58, 63] and used extensively in this work.

2.1. Definition

In the dsTASEP each binding location of a molecular motor on its track is represented by a site $k \in \{1, \ldots, L\}$ of a one-dimensional lattice of L sites. We are not interested in effects due to initiation and termination and therefore we take periodic boundary conditions which makes the model exactly solvable. We ignore the size and mechanochemical cycle of the motor during a translocation step so that a motor is represented by a particle without further internal degrees of freedom. The steric hindrance between motors—which is tantamount to an excluded-volume interaction—leads us to posit that each site can be occupied by one motor or be empty. Thus we introduce local occupation numbers $\eta_k \in \{0, 1\}$ and the complementary 'hole occupation numbers' $\bar{\eta}_k := 1 - \eta_k$. The set of all possible locations of the motors (which is the set of all possible states of the dsTASEP) is denoted by $\boldsymbol{\eta} = (\eta_1, \ldots, \eta_L)$.

The translocation of the motors is thus given by the time evolution of the occupation numbers for which we choose a mathematically convenient discrete-time sublattice dynamics as follows (figure 1). Each discrete time step $t \in \mathbb{N}_0$ corresponds to a mechanochemical cycle of mean time τ between consecutive translocations that depends on the molecular motor under consideration. At even times $t = 2n$, all particles located on odd sites $2k - 1$ move forward from $2k - 1$ to $2k$, provided that site $2k$ is empty. Then, at the following odd time step $t = 2n + 1$, particles move in the same fashion from the even sites $2k$ to $2k + 1$, except across the defect bond $(L, 1)$ where a particle on site L jumps *randomly* to site 1 with probability $0 < p < 1$ provided site 1 is vacant[2]. We mention that the qualitative stationary features of this Markovian stochastic sublattice dynamics, for which the exact stationary distribution is known [33, 63], are the same as for similar parallel and sequential dynamics [64, 65].

With the binary independently and identically distributed random variables $\xi(t)$ with distribution $f(\cdot) = (1 - p)\delta_{\cdot,0} + p\delta_{\cdot,1}$ the two-step stochastic time evolution is thus mathematically defined by the discrete Langevin-type equations

$$\left.\begin{aligned} \eta_{2k-1}(t+1) &= \eta_{2k-1}(t)\eta_{2k}(t) \\ \eta_{2k}(t+1) &= 1 - \bar{\eta}_{2k-1}(t)\bar{\eta}_{2k}(t) \end{aligned}\right\} \quad t \text{ even}, \quad 1 \leqslant k \leqslant \frac{L}{2} \tag{2.1}$$

and

$$\left.\begin{aligned} \eta_{2k}(t+1) &= \eta_{2k}(t)\eta_{2k+1}(t) \\ \eta_{2k+1}(t+1) &= 1 - \bar{\eta}_{2k}(t)\bar{\eta}_{2k+1}(t) \end{aligned}\right\} \quad t \text{ odd}, \quad 1 \leqslant k \leqslant \frac{L}{2} - 1$$

$$\left.\begin{aligned} \eta_L(t+1) &= \eta_L(t)\,[1 - \xi(t+1)\bar{\eta}_1(t)] \\ \eta_1(t+1) &= \eta_1(t) + \xi(t+1)\bar{\eta}_1(t)\eta_L(t) \end{aligned}\right\} \quad t \text{ odd}. \tag{2.2}$$

Translation invariance of the dynamics is broken for $p < 1$. We take $L/2$ even and $0 \leqslant N \leqslant L/2$ particles, corresponding to the density range $\rho := N/L \leqslant 1/2$. Due to the

[2] Notice a slight deviation from the convention of [57] where a full update cycle is defined to be one time step of duration τ with two sub-steps. The limiting cases $p = 0$ (full blockage) and $p = 1$ (no defect) are trivial [33] and excluded from consideration.

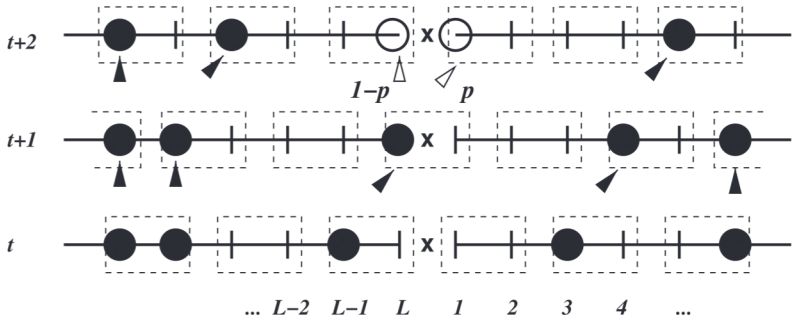

Figure 1. Update scheme of the dsTASEP on a lattice of L sites, illustrated near the slow site L which is indicated by the symbol x between site L and site 1. The particle motion at times t and $t+1$ is shown by arrows, starting with even t. At the slow site the particle jumps with probability p and stays with probability $1-p$. At time $t+2$ the position of the particle which is at site 6 at time $t+1$ is determined by the unspecified occupancy of site 7 at time $t+1$ and therefore not indicated.

particle–hole symmetry $\eta_k \mapsto \bar{\eta}_{L+1-k}$ for all k the properties of the model in the regime $\rho > 1/2$ follow immediately.

Next we introduce the local currents

$$j_{2k-1}(t) := \frac{1}{2}\eta_{2k-1}(t)\bar{\eta}_{2k}(t), \quad 1 \leqslant k \leqslant \frac{L}{2} \tag{2.3}$$

$$j_{2k}(t) := \frac{1}{2}[1-\bar{\eta}_{2k-1}(t)\bar{\eta}_{2k}(t)][1-\eta_{2k+1}(t)\eta_{2k+2}(t)], \quad 1 \leqslant k \leqslant \frac{L}{2}-1 \tag{2.4}$$

$$j_L(t) := \frac{1}{2}\xi(t)[1-\bar{\eta}_{L-1}(t)\bar{\eta}_L(t)][1-\eta_1(t)\eta_2(t)]. \tag{2.5}$$

For even t the local current $j_k(t)$ takes value $1/2$ if between time step $t+2$ and time step t a particle has jumped across the lattice $(k, k+1)$ and 0 otherwise. Hence the quantity $2j_k \in \{0,1\}$ indicates whether or not a jump has taken place locally in a full update cycle of two consecutive time steps. A full update cycle of the dsTASEP can thus be expressed as the discrete continuity equation

$$\frac{1}{2}\left[\eta_k(t+2)-\eta_k(t)\right] = j_{k-1}(t) - j_k(t) \tag{2.6}$$

for all $k \in T_L$ and t even.

2.2. Stationary matrix product measure (MPM)

The invariant measure $P_{L,N}(\boldsymbol{\eta})$ of the dsTASEP (2.1) and (2.2) with a fixed number N of particles—corresponding to a canonical ensemble—was first derived in [33] in terms of a set of rules. There is a critical density $\rho_c = p/2$ where a phase transition from a macroscopically homogeneous phase for $\rho < \rho_c$ to a phase-separated state for $\rho > \rho_c$ occurs. Later a grand-canonical invariant measure $P_L(\boldsymbol{\eta}) \propto \sum_{N=0}^{L/2} z^N P_{L,N}(\boldsymbol{\eta})$ with fluctuating particle number with